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**摘要:** 目的 观察红色毛癣菌刺激人角质形成细胞后γ-IFN及IL-8浓度的变化,以及TLR2对γ-IFN和IL-8分泌的影响。方法 用红色毛癣菌悬液分别刺激TLR2抗体处理前后的角质形成细胞,采用ELISA方法检测不同时间点细胞上清液中γ-IFN及IL-8的浓度,并设置阴性对照;比较TLR2抗体处理前后γ-IFN及IL-8浓度的变化。结果 红色毛癣菌刺激角质形成细胞后,γ-IFN及IL-8浓度明显升高( $P<0.05$ ),4 h即开始,至16 h达高峰;用TLR2抗体中和TLR2后,上清液中IL-8的浓度在2 h、4 h、8 h、16 h各时间点较中和前低,差异有统计学意义( $P<0.05$ );γ-IFN的浓度2 h、4 h、8 h时间点较中和前低,差异有统计学意义( $P<0.05$ ),而在16 h时间点,上清液中γ-IFN的浓度与中和前比较略低,但差异没有统计学意义( $P>0.05$ )。结论 红色毛癣菌刺激角质形成细胞后,可促进角质形成细胞分泌γ-IFN和IL-8;TLR2在角质形成细胞分泌γ-IFN和IL-8的过程中发挥重要的调节作用。

关键词: 角质形成细胞 红色毛癣菌 TLR2 γ-IFN IL-8

**Effects of TLR2 on IL-8 and γ-IFN secretion of keratinocytes in *Trichophyton rubrum* infection**TU Sheng-an<sup>1</sup>, ZHANG Yong<sup>2</sup>, CHEN Hui<sup>2</sup>, CHEN Xing-ping<sup>2</sup>1. Department of Dermatology, Jiujiang University Hospital, Jiujiang 332000, China;  
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**Abstract:** Objective Observe the change of γ-IFN and IL-8 secreted by keratinocytes after *Trichophyton rubrum* infection, and the effects of TLR2 on γ-IFN and IL-8 secretion. To explore the role of TLR2 in anti-dermatophytic infection. Methods Keratinocytes treated before and after anti-TLR2 monoantibody were stimulated by *Trichophyton rubrum*. The levels of γ-IFN and IL-8 in supernatant were determined with ELISA at different time points. Results The levels of γ-IFN and IL-8 secreted by keratinocytes were significantly increased after induced with *Trichophyton rubrum* ( $P<0.05$ ). The level of γ-IFN reached ( $85.36\pm4.54$ ) pg/mL at the point of 4 h, and ( $445.58\pm13.99$ ) pg/mL at 16 h; After treated with anti-TLR2 monoantibody, the levels of IL-8 were reduced at the points of 2 h, 4 h, 8 h and 16 h ( $P<0.05$ ); and the levels of γ-IFN were reduced at the points of 2 h, 4 h and 8 h ( $P<0.05$ ), but had no statistically significant reduction at the point of 16 h ( $P>0.05$ ). Conclusion After stimulated keratinocytes, *Trichophyton rubrum* can promote its secretion of γ-IFN and IL-8; TLR2 play an important role on γ-IFN and IL-8 secretion of keratinocytes.

Keywords: keratinocyte *Trichophyton rubrum* TLR2 γ-IFN IL-8

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